THE RECOVERY KERNEL

Substance-Specific Navigation Series

HOW TO RECOVER FROM ALCOHOL

Using the Recovery Kernel Framework

Alcohol Use Disorder · Heavy Drinking · Beer / Wine / Spirits · Alcohol-Medication Dependence

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE | Crisis: call 911 or text 988

ALCOHOL WITHDRAWAL CAN BE FATAL. READ SECTION 3 FIRST.

A Note Before You Begin

This guide opens with something no other Recovery Kernel navigation guide has opened with: a medical warning that must be read before anything else.

Alcohol withdrawal can kill you. Not as a metaphor. Not as a statistical abstraction. It can produce seizures, cardiac arrhythmias, and a syndrome called delirium tremens that carries a mortality rate of up to 15% without medical treatment. This is not true of cannabis withdrawal or stimulant withdrawal or even most opioid withdrawal. It is specifically, distinctly true of alcohol — and of benzodiazepines, which operate through the same mechanism.

If you are a heavy, daily drinker — meaning you drink to function, drink in the morning, experience shaking or sweating or nausea when you go without alcohol for several hours — you must not stop abruptly without medical supervision. The Recovery Kernel framework in this guide fully applies to your recovery. But the first step is not a supplement stack. It is a phone call to a physician or an urgent care clinic or a detox facility.

For moderate to heavy drinkers without physical dependence — those who drink problematically but do not drink to prevent physical withdrawal — the guide below applies more directly. The distinction is clinical and important. If you are not sure which category you fall into, assume the more cautious one and seek medical assessment.

⚠ IF YOU ARE PHYSICALLY DEPENDENT ON ALCOHOL — STOP AND READ THIS NOW

- Do you drink in the morning to stop shaking?
- Do you experience sweating, rapid heartbeat, or nausea when you go without alcohol for several hours?
- Have you ever had a seizure or hallucinations when stopping or reducing alcohol?
- Have you been drinking heavily and daily for weeks, months, or years?

If you answered yes to any of the above: do not stop drinking abruptly. Call your doctor, go to urgent care, or contact an alcohol detox facility before reducing or stopping. This is not a recommendation. It is a medical necessity. Medically supervised alcohol detox with benzodiazepine tapering is standard of care. It keeps people alive. Please use it.

Once you are medically stable — whether through supervised detox or because you have confirmed you are not in the physical dependence category — the full Recovery Kernel framework below is designed for exactly your biology and your recovery journey.

Section 1 — What Alcohol Did to Your Brain

THE CENTURION — The Most Socially Sanctioned Neurotoxin in Human History

Alcohol is unique among common substances of abuse in that it does not operate through a single receptor system. It touches nearly every major neurotransmitter system in the brain simultaneously — which is why its withdrawal syndrome is more complex and more dangerous than almost any other substance, and why its recovery requires the most comprehensive biological approach.

Understanding what alcohol actually did — not as moral failure but as pharmacology — is the foundation of everything that follows.

The Five Systems Alcohol Disrupted

System	What alcohol did	What withdrawal and recovery look like
GABA (the brake)	Potentiated GABA-A receptors — amplified the brain's primary inhibitory system. Alcohol is a powerful GABA-A agonist. The calm, the reduced anxiety, the social ease, the sleep — all GABA.	With chronic use, GABA receptors downregulate to compensate. When alcohol is removed, the brake system is depleted while the accelerator runs unchecked. Result: seizure risk, tremors, extreme anxiety, insomnia. This is the medical emergency of alcohol withdrawal.
Glutamate (the accelerator)	Inhibited NMDA glutamate receptors — reduced excitatory signaling. The opposite of what stimulants do. Alcohol quiets the excitatory system.	Chronic alcohol use causes glutamate receptor upregulation — the brain compensates for the inhibition by making more accelerators. Remove the alcohol: GABA is depleted, glutamate is upregulated. The brain runs at dangerously high excitation. Seizures are the extreme expression of this imbalance.
Dopamine (the reward signal)	Indirectly increased dopamine release in the nucleus accumbens — the reward center. This is secondary to GABA/glutamate effects but is why alcohol feels good, not just calm.	Dopamine system downregulates similarly to stimulants, but more slowly. Anhedonia, low motivation, and depression in alcohol recovery reflect dopamine system depletion alongside the GABA deficit. The pleasure system is also offline, not just the calm system.
Serotonin	Acutely increases serotonin release — contributing to mood elevation in low doses. Chronic heavy use	Serotonin deficit in recovery produces depression, anxiety, irritability, and sleep disruption. 5-HTP, 5-MTHF, and tryptophan-rich nutrition become critical for serotonin system reconstruction. Often

	depletes serotonin system function.	misidentified as primary depression when it is a recovery-phase deficit.
Opioid system	Triggers endogenous opioid release — beta-endorphins — contributing to the warm social connectedness, pain relief, and euphoric quality of alcohol, especially in OPRM1 A118G carriers.	Endogenous opioid depletion in recovery produces emotional pain sensitivity, difficulty with social bonding, and a specific kind of longing that is often misinterpreted as craving for alcohol when it is actually craving for the endorphin state alcohol produced. OPRM1 genotype determines naltrexone response.

The Acetaldehyde Problem — Unique to Alcohol

THE CENTURION — The Poison Your Liver Is Trying to Clear

Alcohol is metabolized in the liver through two steps: first to acetaldehyde (by the enzyme ADH1B), then to acetate (by the enzyme ALDH2). Acetaldehyde is directly toxic — it damages DNA, proteins, and cell membranes, and is responsible for the flushing, nausea, and rapid heartbeat of the hangover.

This pathway is genetically variable in ways that profoundly affect alcohol's impact and risk profile:

Genetic variant	The biology	What this means for your recovery
ADH1B fast variant (rs1229984)	Converts alcohol to acetaldehyde very rapidly — the toxic intermediate accumulates faster than ALDH2 can clear it.	Fast ADH1B + slow ALDH2 = miserable hangover experience that paradoxically confers some protection against heavy drinking. Fast ADH1B + fast ALDH2 = efficient processing but higher consumption capacity. Know your combination.
ALDH2*2 variant (rs671) — the Asian flush gene	Reduces or eliminates the enzyme that clears acetaldehyde. Heterozygous (*1/*2): ~50% activity, flushing reaction. Homozygous (*2/*2): near-zero activity, extreme toxicity.	Homozygous ALDH2*2 makes alcohol consumption physiologically dangerous — acetaldehyde accumulates to toxic levels with every drink. This variant is protective against AUD but does not prevent it; some people drink through the severe discomfort and develop AUD anyway, with compounded organ damage.
ALDH2*1/*1 (wild type)	Full acetaldehyde clearance capacity — the most common variant in European populations.	No physiological barrier to alcohol consumption from this pathway. Other genetic vulnerabilities

		(GABRA2, OPRM1, COMT) determine addiction risk. Liver damage from chronic heavy use still occurs regardless of efficient ALDH2 — quantity consumed determines hepatotoxicity.

What Chronic Alcohol Did to Your Body — Beyond the Brain

THE CENTURION — The Whole System Was Involved

Alcohol is not merely a brain drug. Years of heavy drinking produce systemic effects that must be addressed in recovery alongside the neurological reconstruction:

- Liver: Fatty liver → alcoholic hepatitis → cirrhosis is the progression with unchecked heavy use. Even without cirrhosis, liver function is often significantly impaired, affecting the metabolism of every supplement and medication in this guide. Liver function tests (LFTs) should be assessed at the start of recovery.
- Thiamine (B1) depletion: Alcohol blocks thiamine absorption. Severe thiamine deficiency produces Wernicke's encephalopathy — a neurological emergency involving confusion, eye movement abnormalities, and loss of coordination. If you have been a heavy drinker and experience any of these symptoms, go to an emergency room immediately. High-dose thiamine (B1) is the treatment. This is not a supplement protocol situation — it is a medical emergency.
- Magnesium depletion: Alcohol dramatically increases urinary magnesium excretion. Magnesium deficiency worsens anxiety, sleep disruption, cardiac arrhythmia risk, and seizure threshold — all of which are already elevated in alcohol withdrawal. Magnesium repletion is the highest-priority supplement intervention in alcohol recovery.
- Gut and microbiome devastation: Alcohol destroys the intestinal barrier, disrupts the microbiome, and produces intestinal permeability ('leaky gut') that drives systemic inflammation, mood disruption, and craving. The gut-brain axis (Protocol 09 and Centurian Paper 18 in the main Compendium) is a central repair target.
- Cardiovascular: Chronic heavy drinking produces cardiomyopathy, arrhythmia (particularly atrial fibrillation), and elevated blood pressure. Cardiovascular assessment should be part of early recovery medical evaluation.
- Immune suppression: Alcohol chronically suppresses immune function. Many people in early alcohol recovery are immunologically depleted. Nutrition and sleep are the primary immune repair tools.

Section 2 — Your Genomic Blueprint

Alcohol use disorder has one of the highest heritability estimates of any condition in medicine — approximately 50–60%. This is not destiny. It is biology that can be understood, worked with, and addressed. The Recovery Kernel's genomic framework reveals which specific biological systems made alcohol feel necessary, and which ones most urgently need support in recovery.

The DNA Testing Panel for Alcohol Recovery

Gene	What it controls	Key variant	Alcohol recovery relevance
GABRA2	GABA-A receptor alpha-2 subunit — inhibitory tone baseline	rs279858 / rs279845 high-risk haplotype	THE most important alcohol-specific gene in the Recovery Kernel framework. High-risk variants structurally reduce baseline GABAergic inhibitory tone — the brain's brake system runs at chronically low power. Alcohol powerfully activates this underpowered system, producing relief that is genuinely extraordinary for this genotype. Recovery must build alternative GABA support — magnesium, GABA-supporting practices, stress reduction — as first-line priorities.
OPRM1	Mu-opioid receptor — endorphin response and social bonding reward	A118G (rs1799971)	A118G carriers experience amplified dopamine release in the nucleus accumbens in response to alcohol — a stronger reward signal than the A/A genotype. This is the strongest predictor of naltrexone response in alcohol use disorder. If you carry A118G, naltrexone is likely to be significantly more effective for you than for the average patient. Bring this to your physician.
COMT	Dopamine and stress chemical clearance speed	Val158Met (rs4680)	Slow COMT (Met/Met, Sage constitution) is heavily represented in alcohol use disorder — the chronically anxious brain that cannot quiet itself finds alcohol's GABA potentiation to be the most effective off switch available. Anxiety management is the central recovery strategy for this profile. Fast COMT

			(Warrior) also appears — social drinkers seeking dopaminergic stimulation from alcohol's secondary dopamine effects.
ALDH2	Aldehyde dehydrogenase 2 — acetaldehyde clearance	rs671 (*1/*1, *1/*2, *2/*2)	Critical for understanding both risk profile and body damage burden. *2/*2 homozygous individuals who drank despite extreme discomfort have significant acetaldehyde-mediated organ damage that standard recovery protocols must account for. Liver function testing is mandatory. N-acetyl cysteine and liver support supplements take on additional urgency.
ADH1B	Alcohol dehydrogenase — converts alcohol to acetaldehyde	rs1229984 (fast vs. slow variant)	Fast ADH1B (His48 allele) produces acetaldehyde faster — conferring some natural deterrence but also more rapid toxic exposure if drinking continues. Slow ADH1B allows higher alcohol consumption before acetaldehyde accumulation — associated with higher AUD risk in some populations.
MTHFR	Methylation — foundation of all neurotransmitter synthesis	C677T (rs1801133), A1298C (rs1801131)	Alcohol depletes folate and disrupts methylation actively. In MTHFR variant carriers, this compounds an existing deficit — producing severe neurotransmitter synthesis impairment. 5-MTHF and methylcobalamin supplementation are highest priority and often produce dramatic early recovery improvement in this profile.
DRD2 / ANKK1	Dopamine D2 receptor density	Taq1A (rs1800497)	A1 allele (reduced D2 density) is associated with alcohol use disorder through the Reward Deficiency Syndrome pathway. Alcohol provides dopaminergic relief for the reward-deficient brain. Recovery must build multiple dopamine sources — this profile cannot be expected to find sobriety satisfying without deliberate reward architecture.
SLC6A4 / 5-HTTLPR	Serotonin transporter expression	Short/short allele	Short/short carriers have higher anxiety sensitivity and higher AUD risk — particularly for the anxious-drinking pattern. Serotonin reconstruction through 5-HTP, 5-MTHF, and tryptophan-rich nutrition addresses the biological vulnerability

			directly. SSRIs may be appropriate depending on severity — discuss with physician.
CYP2E1	Cytochrome P450 2E1 — metabolizes alcohol in the liver; generates reactive oxygen species	Several polymorphisms (rs2031920, rs3813867)	High CYP2E1 inducibility increases oxidative stress from alcohol metabolism — worsening liver damage and neuroinflammation. N-acetyl cysteine (NAC) and antioxidant support (glutathione precursors, alpha-lipoic acid) are particularly important for this profile.
CYP2C19 / CYP2D6	Metabolism of many medications used in alcohol recovery	Poor to ultra-rapid metabolizer variants	Affects metabolism of naltrexone (CYP3A4), acamprosate (not CYP metabolized — renal), disulfiram, and many psychiatric medications used in co-occurring condition treatment. Poor metabolizers experience higher drug levels; ultra-rapid may get insufficient effect. Essential for medication optimization.

Phenotyping Without Test Results

The compendium's phenotyping tools apply directly here. Ask yourself:

- Did alcohol primarily calm you — reduce anxiety, quiet the mind, make social situations manageable? → Likely Sage COMT (Met/Met) + GABRA2 high-risk. Anxiety is your primary recovery target.
- Did alcohol make you feel alive, social, confident, and stimulated? → Social-reward pattern, likely OPRM1 A118G + possible Warrior COMT. Endorphin system and dopamine support are priority.
- Do you have a family history of alcohol use disorder across multiple generations? → DRD2 deficit and GABRA2 variants likely. RDS is probably a significant component.
- Did alcohol cause you more physical discomfort (flushing, rapid heartbeat) than others seemed to experience? → Possible ADH1B fast variant or ALDH2 variant. Liver support is elevated priority.
- Did antidepressants never quite work for you? Did folate supplements seem to help? → Likely MTHFR involvement. Start 5-MTHF immediately.

Section 3 — The Detox Protocol

⚠ READ THIS SECTION BEFORE ANY OTHER ACTION IF YOU ARE CURRENTLY DRINKING

3.1 — Medical Detox: When It Is Required

THE CENTURION — The Line That Cannot Be Crossed Without Medical Support

Alcohol withdrawal is the only common substance withdrawal where the following sequence is possible, documented, and occurs without warning: anxiety → tremor → agitation → seizure → delirium tremens → death. This sequence can begin within 6–48 hours of the last drink in a physically dependent person.

Medical detox is required — not recommended, required — if you meet any of the following criteria:

- You drink daily and have done so for more than 2–3 weeks
- You experience any physical withdrawal symptoms between drinks: shaking, sweating, racing heartbeat, nausea, anxiety that alcohol relieves
- You have ever had a seizure during alcohol withdrawal
- You have ever had delirium tremens — confusion, hallucinations, fever, extreme agitation
- You are also using benzodiazepines or other GABAergic substances — combined withdrawal is especially dangerous
- You have a GABRA2 high-risk variant — if you know this, inform your detox team

What Medical Detox Looks Like

Outpatient detox: appropriate for mild-to-moderate physical dependence with a safe home environment and daily medical contact. Benzodiazepine taper (typically diazepam or chlordiazepoxide) prescribed to prevent seizures while the brain recalibrates.

Inpatient detox: required for severe dependence, prior seizure history, GABRA2 high-risk profile, co-occurring medical conditions, or absence of safe home environment. 3–7 days with continuous monitoring.

The CIWA protocol: Clinical Institute Withdrawal Assessment for Alcohol — the standard tool medical teams use to assess and manage withdrawal severity. Ask your detox team about it. Understanding it helps you communicate your symptoms accurately.

⚠ NEVER SUBSTITUTE SUPPLEMENTS FOR MEDICAL DETOX IN PHYSICAL DEPENDENCE. SUPPLEMENTS DO NOT PREVENT WITHDRAWAL SEIZURES.

The supplement and peptide protocol in this guide begins after medical stabilization — not instead of it. If you are not physically dependent, or once you have completed medical detox, the biological reconstruction framework below applies fully.

3.2 — The Withdrawal Timeline: Post-Medically Supervised Detox

THE CENTURION — What the Brain Is Moving Through

Timeline	Biology	Symptoms	What to do
Hours 6–24 (if not medically supervised — seek help)	GABA systems failing as alcohol clears; glutamate systems surging unchecked	Tremor, sweating, anxiety, rapid heartbeat, nausea, insomnia	THIS IS THE MEDICAL WINDOW. Call a physician, go to urgent care, or call 911. Do not manage this alone if you are physically dependent.
Hours 24–72 (peak medical risk)	Maximum GABA-glutamate imbalance; cortisol surging; autonomic nervous system dysregulated	Severe anxiety, confusion, possible hallucinations, seizure risk	Medical supervision required for physically dependent individuals. If medically supervised: benzodiazepine protocol managing the GABA gap. Begin high-dose thiamine (B1) immediately — Wernicke's prevention is urgent.
Days 3–7 (stabilization)	Acute medical risk passing; GABA systems beginning to rebound; glutamate calming; but significant neurochemical instability remains	Profound fatigue, emotional rawness, sleep disruption, depression, cognitive fog, strong cravings	Begin supplement stack (Section 4). Sleep is the priority — the recovering brain desperately needs it. Begin nutrition protocol — the depleted system needs rebuilding material. Allow the fatigue.
Weeks 1–4 (The Rebuilding)	GABA receptor density recovering; glutamate normalizing; dopamine and serotonin systems beginning to repair; liver starting to	Intermittent anxiety, depression, sleep difficulty, mood volatility, cravings that come in waves rather than continuously	Full supplement protocol running. Exercise begins — BDNF is the repair signal. PAWS episodes likely — see Section 3.3. Begin protocol navigation (Section 7).

	recover function		
Months 1–6	Significant GABA receptor recovery; serotonin and dopamine reconstruction; liver healing if drinking has stopped; brain volume beginning to increase (alcohol causes measurable brain atrophy that reverses in sobriety)	Progressive improvement with episodic difficulty; cognitive clarity returning; emotional range widening; sleep normalizing; physical health measurably improving	This is when sobriety starts to feel like something rather than merely the absence of something. The brain is rebuilding itself in ways visible on MRI. The work done now determines the quality of the next decade.
Months 6–24+	Brain volume substantially restored; GABA and glutamate systems normalized; dopamine and serotonin baseline recovered; liver health largely restored in most without cirrhosis	Near-normal emotional regulation, cognition, and pleasure; episodic cravings primarily contextual; genuine enjoyment of sober life accessible	The recovered brain at 12–24 months of sobriety shows measurable reversal of the gray matter loss produced by chronic drinking. This is one of the most compelling biological arguments for sustained sobriety that exists.

3.3 — Post-Acute Withdrawal Syndrome in Alcohol Recovery

⚠ PAWS EPISODES CAN LAST UP TO 2 YEARS. THIS IS NOT RELAPSE. IT IS NEUROBIOLOGY.

Post-acute withdrawal syndrome in alcohol recovery is particularly prolonged and can be severe. GABA and glutamate systems that were chronically imbalanced do not simply normalize and stay normalized — they oscillate during the recovery period, producing episodes of acute anxiety, depression, insomnia, and craving that can feel indistinguishable from early withdrawal.

PAWS is most commonly triggered by: stress, poor sleep, illness, grief, conflict, high-sugar food, caffeine, and exposure to alcohol cues. The GABRA2 high-risk genotype is associated with more severe and prolonged PAWS.

When a PAWS episode hits: return to Protocol 01 (Shelter) in the main Compendium. Increase magnesium. Call your support structure. Do not interpret the episode as evidence that recovery is not working. It is the nervous system calibrating, not failing.

Section 4 — Biological Reconstruction

This section addresses the simultaneous reconstruction of five systems that alcohol disrupted: GABA/glutamate balance, dopamine, serotonin, the opioid/endorphin system, and the methylation foundation that underlies all neurotransmitter synthesis. These are not sequential targets — they are addressed in parallel, with priorities determined by your specific genomic profile and clinical presentation.

Everything in this section: discuss with your physician or pharmacist before starting, especially given the liver involvement common in alcohol recovery. Some supplements are contraindicated in liver disease.

4.1 — The Emergency Nutritional Replenishment

THE CENTURION — The Body Was Starved While the Drinking Continued

Chronic heavy alcohol use produces a specific nutritional crisis that must be addressed before anything else in the recovery supplement stack can work effectively. The depleted brain cannot synthesize neurotransmitters without precursors. The precursors cannot be converted without cofactors. The cofactors were systematically destroyed by years of drinking.

Nutrient	Priority level	Target dose	Why this specific nutrient is critical in alcohol recovery
Thiamine (Vitamin B1)	CRITICAL — Day 1	100–500mg daily	Wernicke's encephalopathy prevention. Alcohol blocks thiamine absorption — severe deficiency produces irreversible neurological damage. If any confusion, memory problems, or eye movement abnormalities are present, emergency high-dose IV thiamine is required — go to an ER. For all others: oral thiamine is the highest-priority supplement on Day 1 of recovery.
Magnesium glycinate	CRITICAL — Day 1	400–600mg elemental daily	Alcohol dramatically increases urinary magnesium loss. Magnesium deficiency worsens seizure threshold, cardiac arrhythmia risk, anxiety, and insomnia — all already elevated in alcohol recovery. Glycinate form for best absorption and neurological effect. Split morning and evening dosing.
B-Complex (full methylated)	High — Day 1	Full B-complex	Alcohol depletes the entire B-vitamin spectrum: B1, B2, B3, B5,

		with methylated forms	B6, B9, B12. Use a B-complex that includes methylated forms (methylcobalamin, methylfolate) — not cyanocobalamin or folic acid. The depleted methylation system cannot convert the inactive forms.
5-MTHF (L-methylfolate)	High — Day 1	400–1000mcg daily	Alcohol specifically depletes folate, and standard folic acid supplementation does not address MTHFR-variant methylation impairment. 5-MTHF bypasses the MTHFR enzyme entirely and provides activated folate directly to neurotransmitter synthesis pathways. Often produces noticeable mood improvement within 1–2 weeks.
Zinc	High — Day 1	25–30mg daily with food	Alcohol reliably depletes zinc. Zinc is required for GABA receptor function, dopamine synthesis, immune function, and wound healing. Alcohol recovery places all of these systems under demand simultaneously. Zinc is the enabling mineral for this multi-system repair.
Vitamin D3 + K2	High — Week 1	3000–5000 IU D3 + 100mcg K2 daily	Heavy drinkers are almost universally deficient in Vitamin D. D3 regulates gene expression, mood, immune function, and GABA synthesis. K2 ensures D3-mobilized calcium is directed to bones rather than arteries. Take with a fatty meal for absorption.
Vitamin C	Moderate — Week 1	500–2000mg daily, divided	Potent antioxidant that addresses the oxidative stress of alcohol withdrawal. Also supports collagen synthesis (alcohol damages skin, gut lining, and connective tissue), immune function, and dopamine synthesis (cofactor for dopamine beta-hydroxylase).
Protein and amino acid support	High — Day 1	0.8–1g per lb body weight daily	Alcohol suppresses appetite and provides empty calories that crowd out protein. The depleted amino acid pool cannot synthesize neurotransmitters regardless of cofactor availability. Protein is the raw material. Target 30–50g per meal. Eggs, fish, chicken, Greek yogurt, legumes. If appetite is

			suppressed, protein shakes are appropriate.

4.2 — GABA System Restoration

THE CENTURION — Rebuilding the Brake System That Was Borrowed Against for Years

The GABA system does not replenish through supplementation the way the dopamine system can be partially supported with precursors. GABA receptor upregulation is a structural process that occurs through abstinence and time. What supplements can do is support the conditions that allow this upregulation to proceed safely and effectively.

Supplement / approach	Dose	Timing	Role in GABA recovery
Magnesium glycinate (repeated)	300–400mg elemental	Morning and evening	The most important GABA-supporting supplement. Magnesium is an NMDA antagonist — it reduces glutamate excitotoxicity — AND it is required for GABA synthesis AND it supports GABA-A receptor function. It addresses the GABA/glutamate imbalance from both sides simultaneously. There is no better single supplement for the alcohol-withdrawal nervous system.
L-Theanine	200–400mg	As needed, especially PM	Increases GABA and glycine; promotes alpha-brainwave state. Non-sedating. Does not produce dependence. Provides genuine calming without the addiction liability of benzodiazepines, which is critical in alcohol recovery where benzo substitution is a documented relapse trap.
Taurine	500–2000mg daily	With meals	Taurine is a GABA-mimetic amino acid that acts at GABA-A receptors. Alcohol depletes taurine significantly. Taurine supplementation in alcohol recovery has been studied with positive effects on withdrawal anxiety and

			hepatoprotection. A dual-action supplement: calms the nervous system AND protects the liver.
Inositol	2–12g daily (titrate up)	Divided doses with food	Modulates GABA and serotonin receptor sensitivity. High-dose inositol has evidence for anxiety and OCD — both common in alcohol recovery. Supports the insulin signaling pathways disrupted by alcohol-induced blood sugar dysregulation.
GABA (direct supplement)	250–750mg	Evening	Controversial: GABA itself may not cross the blood-brain barrier efficiently. However, peripheral GABA receptors and gut GABA activity contribute to calming, and some clinical experience supports its use in anxiety. Phenibut (a GABA derivative) is NOT recommended — it is addictive and substitutes one GABAergic dependence for another.
Lemon balm extract (Melissa officinalis)	300–600mg standardized	Evening	GABA transaminase inhibitor — slows GABA breakdown, increasing available GABA. Gentle anxiolytic effect. One of the safer herbal GABA-supporting options without addiction liability. Useful in the weeks 2–8 window when anxiety is the primary challenge.

4.3 — Serotonin and Dopamine Reconstruction

Once the nutritional emergency is addressed and the GABA system is being supported, serotonin and dopamine reconstruction become the central biological targets. These systems were chronically disrupted by alcohol and are the substrate for the depression, anhedonia, and motivational deficits that drive relapse in alcohol recovery.

Supplement	Dose	Timing	Alcohol-specific notes
5-HTP (serotonin precursor)	50–200mg	Evening — 30 min before bed	Serotonin is depleted by chronic alcohol use. 5-HTP

			provides the direct precursor. Start at 50mg — if tolerated, increase. Do not combine with SSRIs or SNRIs without physician guidance (serotonin syndrome risk). Do not take in the morning if mood elevation causes anxiety. Evening dosing supports serotonin conversion to melatonin for sleep.
L-Tryptophan	500–2000mg	Evening, away from protein	An alternative or complement to 5-HTP — slightly slower-acting as it requires the additional conversion step. May cause less overshoot than 5-HTP in sensitive individuals. Combined tryptophan + 5-HTP at lower doses of each is an option for those who find either alone too stimulating.
L-Tyrosine / NALT	500–1500mg	Morning, away from protein	Dopamine precursor. Useful for the anhedonia and motivational deficit of alcohol recovery — particularly in Warrior COMT or DRD2-deficit profiles where dopamine support is high priority. Start after Day 7 when the acute phase has settled. Do not use with MAOIs.
SAMe	400–800mg	Morning, away from food	Universal methyl donor — directly supports dopamine and serotonin synthesis and the methylation pathways that process them. Evidence for depression in alcohol recovery. CAUTION: contraindicated in bipolar disorder; can trigger mania. Check CYP interaction with any medications. Start low.
Rhodiola rosea	300–600mg	Morning	Dopamine and serotonin modulation; adaptogen; anti-fatigue. Mild antidepressant effect with strong evidence base. Particularly useful weeks 2–8 when motivation and energy are low but the precursor stack alone is

			insufficient. Do not take late in the day — activating.

4.4 — Liver Support Protocol

THE CENTURION — The Organ That Processed Every Drop Must Now Heal

⚠ If you have known liver disease, cirrhosis, or significantly elevated LFTs, consult your physician before starting any supplement protocol. Some supplements are hepatotoxic in liver disease despite being safe in healthy liver function.

Supplement	Dose	Timing	Liver-specific role
NAC (N-Acetyl Cysteine)	600–1200mg twice daily	Morning and evening with food	Glutathione precursor — NAC replenishes glutathione, the liver's primary antioxidant and detoxification molecule. Alcohol massively depletes hepatic glutathione. NAC is one of the most evidence-supported liver-protective supplements and simultaneously reduces alcohol craving through glutamate homeostasis. Dual-action essential.
Milk thistle (silymarin)	140–420mg standardized (70–80% silymarin)	With meals	The most studied hepatoprotective botanical. Silymarin has anti-inflammatory, antioxidant, and antifibrotic effects in the liver — directly relevant to alcoholic liver disease at every stage except end-stage cirrhosis. Well-tolerated. One of the few supplements where the evidence in alcohol-induced liver disease is genuinely strong.
Alpha-lipoic acid (ALA)	300–600mg daily	Away from food for best absorption	Both water and fat soluble — reaches compartments other antioxidants cannot. Regenerates glutathione, Vitamin C, and Vitamin E simultaneously. Reduces acetaldehyde toxicity. Particularly relevant for ALDH2 variant carriers who accumulate excess

			acetaldehyde.
TUDCA (tauroursodeoxycholic acid)	250–500mg daily	With food	A bile acid derivative with strong hepatoprotective effects — reduces endoplasmic reticulum stress and mitochondrial dysfunction in liver cells. Emerging evidence in alcoholic liver disease. Expensive but worth consideration for those with significant liver involvement.
Phosphatidylcholine	1–2g daily	With food	Essential phospholipid for liver cell membrane integrity. Alcohol disrupts hepatic phosphatidylcholine synthesis. Supplementation supports membrane reconstruction, bile flow, and overall hepatocyte function. Also provides choline for acetylcholine synthesis — relevant for cognitive recovery.

4.5 — The Craving Interrupt Protocol

THE CENTURION — Alcohol Cravings Are Not Requests. They Are Neurological Events.

Alcohol cravings in recovery operate through at least three distinct neurobiological channels — GABA deficit craving (anxiety-driven), dopamine deficit craving (reward-seeking), and opioid/endorphin deficit craving (social pain and longing). Understanding which channel is activating the craving changes how you respond to it.

Craving type	What it feels like	Immediate interrupt
GABA-deficit craving (the most common in alcohol recovery)	Anxiety-driven. The nervous system is sending an alarm signal. Alcohol quiets alarms. The craving is the nervous system requesting its most reliable alarm-silencer.	4-7-8 breathing immediately — activates the vagus nerve and provides measurable GABAergic relief. L-theanine 200mg. Cold water on face and wrists — activates the dive reflex, immediate parasympathetic activation. Magnesium if not already taken today.
Dopamine-deficit	Flat, gray, joyless. The world	High-intensity exercise — the

craving	has no color. Activities that should feel good do not. The craving is the reward system requesting stimulation.	fastest available dopamine intervention. Cold exposure (shower or plunge). Social contact with someone whose company generates genuine warmth. Execute the highest-intensity item from your natural reward architecture (Section 6).
Opioid/endorphin craving	Lonely. Emotionally raw. Yearning for connection or comfort that feels out of reach. Often misread as craving for alcohol specifically when it is actually craving for the warmth and belonging alcohol's endorphin release was producing.	Human contact — physical if possible (hug, handshake), voice at minimum. Do not isolate with this craving. Exercise also releases endorphins. Laughter. Any activity that produces genuine oxytocin and endorphin co-release: playing with a pet, physical contact with someone safe, vigorous movement with another person.

THE 5-MINUTE CRAVING INTERRUPT — EXECUTE IN ORDER

1. IDENTIFY THE CRAVING TYPE. Which of the three above is this? Anxiety-based, flatness-based, or loneliness-based? Naming it correctly changes the response.
2. EXECUTE THE 4-7-8 BREATH. Four full cycles. No exceptions. This is the universal first step regardless of craving type.
3. TAKE THE APPROPRIATE SUPPLEMENT. Magnesium + L-theanine for GABA craving. L-tyrosine or Rhodiola for dopamine craving (if it's morning and you haven't taken them). Neither replaces steps 1–2 — they support what your body is already doing.
4. CHANGE ONE ENVIRONMENTAL VARIABLE. The craving circuit requires the cue environment. Move location, change what is in your field of vision, go outside.
5. MAKE CONTACT. One text or call. Not to discuss the craving — just to be in connection. Isolation is the environment where alcohol cravings execute.
6. EXECUTE THE NATURAL REWARD. From Section 6. Whatever is highest-intensity and immediately available. You are practicing the redirect — not just resisting, but replacing.
7. DOCUMENT. Trigger, type, intensity (0–10), action, outcome. The documented craving becomes data. Data becomes leverage.

4.6 — Peptide Support Layer

The following peptides have mechanistic relevance to GABA system recovery, neuroinflammation reduction, and liver repair. Research compounds — physician discussion required.

Peptide	Mechanism in alcohol recovery	Timing	Notes
BPC-157	Gut-brain axis repair; neuroplasticity; dopamine and serotonin modulation; anti-inflammatory; direct hepatoprotective effects in research models	Day 1 onwards — daily	BPC-157 is the highest-relevance peptide in alcohol recovery because it simultaneously addresses three of alcohol's primary damage domains: gut barrier (leaky gut), dopamine-serotonin systems, and liver. Oral administration is appropriate and may be superior for gut healing. The gut-brain axis repair may be its most important alcohol recovery function.
Selank	GABA and serotonin modulation; anxiolytic without dependence; reduces anxiety without sedation	Weeks 1–8 — as needed for anxiety; daily if GABA-craving is predominant	Selank is particularly valuable in alcohol recovery because the GABA-deficit anxiety is the primary craving driver and primary relapse risk. Selank addresses this without the addiction liability of benzodiazepines. Non-sedating, non-habit-forming. Intranasal.
Epitalon	Sleep architecture; pineal pathway; telomere protection; circadian rhythm normalization	Weeks 1–4 — 10-day cycle, then as needed	Sleep disruption in alcohol recovery persists for weeks to months. Epitalon supports the pineal pathway that alcohol chronically suppressed. Also provides cellular-level protection during the oxidative stress of early recovery.
Thymosin Beta-4 (TB-500)	Anti-inflammatory; tissue repair; liver fibrosis reduction in research models	Months 1–3 if liver involvement is significant	For those with alcoholic liver disease where fibrosis is present, TB-500 has documented anti-fibrotic effects in research models. This is physician-territory given liver disease complexity. Not first-line but worth discussion with a

			physician managing your liver recovery.
Semax	BDNF upregulation; cognitive repair; prefrontal cortex support; dopamine and serotonin modulation	Weeks 2 onwards	Alcohol produces measurable cognitive impairment and prefrontal cortex thinning. Semax's BDNF-stimulating effects support the neural repair that is occurring during sobriety. Particularly valuable for the cognitive fog and motivational impairment of months 1–3.

Section 5 — Rebuilding Sleep

THE CENTURION — Alcohol Did Not Help You Sleep. It Sedated You. These Are Not the Same Thing.

One of the most common and most dangerous beliefs in alcohol use disorder is that alcohol helps sleep. Alcohol produces sedation — rapid loss of consciousness. It does not produce sleep. It suppresses REM sleep almost entirely, fragments sleep architecture, reduces slow-wave deep sleep, and produces rebound insomnia in the second half of the night as alcohol is metabolized.

The result is that years of 'drinking to sleep' produce a brain that has never experienced genuine restorative sleep while drinking — and now, in early recovery, struggles to sleep at all because the sedation mechanism is gone and the brain has not practiced the natural sleep initiation process for years.

Sleep reconstruction in alcohol recovery is one of the most important and most underestimated aspects of the process. Insomnia is the most common cited driver of early relapse. It is also one of the most responsive targets to the supplement and behavioral protocol below.

- Melatonin (0.5–1mg sublingual, 30–60 min before target sleep time) — low dose is more effective than high dose; high doses disrupt circadian rhythm
- Magnesium glycinate (300–400mg, 1 hour before bed) — supports GABA, reduces glutamate excitotoxicity, lowers core temperature
- Glycine (3–5g, 30 min before bed) — reduces core body temperature, the primary physiological sleep trigger; also NMDA co-agonist that supports the GABA-glutamate rebalancing
- L-Theanine (200–400mg evening) — alpha-wave promotion, reduces the anxious hyperarousal that prevents sleep onset
- 5-HTP (50–100mg evening) — serotonin precursor that converts to melatonin; supports both mood and sleep simultaneously
- Phosphatidylserine (100–200mg evening) — reduces evening cortisol; the HPA axis dysregulation from chronic drinking elevates cortisol and prevents sleep onset

Also execute Protocol 17 (Sleep Architecture) from the main Compendium in full. The alcohol recovery additions: avoid all alcohol including cooking alcohol and mouthwash with alcohol — even trace amounts activate the reward circuit and disrupt sleep architecture in early recovery. The vivid dreams of early recovery are REM rebound — they are healing, not symptoms to suppress.

Section 6 — Porting the Addiction: Finding Your Own GABA

THE CENTURION — Centurian Paper C-28 Applied to Alcohol Recovery

Alcohol provided at least three distinct neurochemical states depending on how, when, and why you used it. Each must be replaced by natural means that do not cost you everything else.

What Alcohol Was Actually Providing

The function	The neurochemistry	The natural replacement architecture
Anxiety relief / nervous system quiet	GABA potentiation — alcohol silenced the alarm system that wouldn't stop. For the Sage constitution with chronically elevated stress chemicals, this was the most powerful relief available.	Breathwork (daily, non-negotiable). Meditation — the contemplative practice that builds the internal GABA system rather than borrowing against an external supply. Cold exposure (produces post-stress calm). Nature. Magnesium. The practice is daily and lifelong — not situational.
Social ease and connection	Endorphin release + reduced social inhibition. Alcohol dissolved the self-consciousness barrier and produced warmth, openness, and the sense of belonging.	The goal is not to become naturally as disinhibited as alcohol made you — it is to build genuine connection that does not require disinhibition. Twelve-step communities, small groups, sober social communities. Physical contact (safe and consensual) releases oxytocin. Service — helping others — produces endorphins and belonging simultaneously.
Pleasure and reward	Dopaminergic and opioid system activation. Alcohol feels good, especially in the early stages. This is real. It was a real neurochemical event.	Multiple dopamine sources built deliberately: vigorous exercise, creative mastery, competitive activity, achievement with social stakes. No single source will replicate alcohol's full-system activation — which is why multiple sources are essential rather than optional.
Transition ritual and permission to rest	The drink after work was not primarily about pharmacology. It was about a signal — the day is done, rest is permitted, the	Replace the ritual precisely: same time, same location, different content. Non-alcoholic ritual drink (sparkling water,

	performance can stop.	herbal tea, non-alcoholic cocktail). Brief ceremony of transition. The brain needs the signal; it does not need the alcohol in the signal.

The Natural GABA Architecture for Alcohol Recovery

THE CENTURION — Building the Internal Brake System the Drug Was Running

The Sage constitution's recovery — and alcohol recovery is predominantly, though not exclusively, a Sage story — requires building a life that provides sufficient nervous system calm through owned means. This is not about eliminating stimulation. It is about building a resilient internal regulation system that does not require a substance to function.

- A daily contemplative practice of minimum 10 minutes. Not as a preference. As a non-negotiable biological necessity. This is what builds the endogenous GABA tone that alcohol was supplementing. Protocol 04 (Dharma Recovery) in the main Compendium is the reference.
- Vigorous aerobic exercise at least 4 times per week. The BDNF release from sustained aerobic exercise is the primary neural repair mechanism for alcohol-damaged brains. The endorphin release addresses the opioid/endorphin system simultaneously. This is not lifestyle advice — it is the most powerful non-pharmacological intervention for alcohol recovery that exists.
- A creative practice with genuine depth. The flow state is the natural GABA state — total absorption in a demanding activity quiets the anxious mind through the mechanism of full attentional engagement rather than chemical suppression. Writing, music, visual art, cooking, building. Depth over breadth.
- Authentic community. Not performance community. Not social media community. In-person connection with people who know your actual situation, with whom the mask is optional. The endorphin and oxytocin system that alcohol was activating responds specifically to this. Twelve-step communities provide this for many people — and the Compendium's Protocol 04 and Protocol 08 address the social architecture of recovery broadly.
- Nature exposure as a scheduled intervention. Not incidentally. Minimum 20 minutes daily. The recovering nervous system's cortisol levels normalize measurably in natural environments in ways they do not in urban or indoor settings.
- Service. The act of genuinely helping another person activates the reward, connection, and meaning circuits simultaneously and provides the sense of being useful in the world that alcohol's social lubricant was partially

producing. Many people in long-term alcohol recovery report that service is the most reliable source of sustained wellbeing they have found.

Section 7 — Navigating the Recovery Kernel: Your Protocol Map

Phase	Protocols	Primary focus	Alcohol-specific notes
Medical Detox (Days 0–7 if physically dependent)	Medical provider — not the Compendium	Survival. Seizure prevention. Medical stabilization.	The Recovery Kernel framework does not begin here. Medical detox begins here. Once medically stable, return to this guide and start at the Biological Foundation phase.
Biological Foundation (Days 1–14)	Protocol 07, Protocol 09, Protocol 17, Protocol 18	Emergency nutrition, thiamine, magnesium, sleep, gut repair	Thiamine on Day 1. Magnesium on Day 1. Full B-complex on Day 1. These are not optional. The depleted system cannot begin recovery without its raw materials. Protocol 18 (nutrition) and Protocol 09 (gut-brain axis) are the highest-priority Volume I protocols for alcohol recovery.
Genomic Mapping (Weeks 1–4)	Section 15 (Compendium), Protocol 11, Protocol 12	GABRA2 assessment, OPRM1 for naltrexone guidance, COMT constitution, MTHFR	The GABRA2 high-risk variant and OPRM1 A118G are the two highest-priority genomic findings in alcohol recovery — they directly determine medical treatment decisions (seizure monitoring, naltrexone appropriateness). Get tested as early as possible.
GABA System Repair (Weeks 1–12)	Protocol 04, Protocol 01 craving interrupt, Section 4.2 of this guide	GABA supplement stack, breathwork, contemplative practice, anxiety architecture	Unlike stimulant or cannabis recovery, GABA system repair is the PRIMARY biological target in alcohol recovery — it is both the most damaged system and the one whose deficit most directly drives relapse. Everything else happens within this container.
Behavioral and Environmental Architecture (Weeks 2–12)	Protocol 02, Protocol 05, Protocol 06, Protocol 08, Protocol 13	Addiction transfer prevention, environmental redesign, trigger mapping, social architecture	Alcohol-to-benzodiazepine transfer is the most medically dangerous addiction transfer in recovery. Protocol 02 must be read immediately. Cannabis transfer is also common for the anxious drinker. Name the transfer risks explicitly before they appear.
Liver and Physical Recovery (Months 1–6)	Protocol 07, Protocol 18, liver support stack (Section 4.4)	Liver function monitoring, NAC, silymarin, TUDCA, phosphatidylcholine	Repeat LFTs at 1 month, 3 months, and 6 months of sobriety. The liver is one of the most regeneratively capable organs in the body — significant recovery is possible even

			from advanced damage with sustained abstinence. Track it. Celebrate the improvement.
Identity and Meaning (Months 2–12)	Protocol 20, Protocol 21, Protocol 23, C-28	Identity reconstruction, the sober self, purpose architecture	Alcohol use disorder often deeply intertwines with identity — 'I am someone who drinks.' For long-term drinkers, sobriety requires constructing an identity that does not have a substance-shaped hole in the middle of it. Protocol 20 (identity reconstruction) is essential.
Long Game (Months 6–24+)	Protocol 24, Protocol 25, C-28, C-31	Purpose architecture, relational repair, the natural addiction fully operational	The brain volume restoration that occurs in the second year of sobriety is measurable on MRI and genuinely significant. The person who committed to recovery at month 1 can be neurologically different — meaningfully, measurably better — by month 18. This is not metaphor. It is the most powerful long-term argument for sobriety that biology provides.

Section 8 — The Medical Support Layer

Alcohol use disorder has the most robust evidence base of any addiction for pharmacological treatment. The medications below are underutilized — studies consistently show that fewer than 10% of people with AUD who could benefit from these medications receive them. This is a treatment gap that costs lives.

Bring this section to your physician. Ask directly about these options. The Recovery Kernel framework and pharmacological treatment are not in competition — they are designed to work together.

FDA-Approved Medications for Alcohol Use Disorder

Medication	Mechanism	Evidence level	Genomic and clinical considerations
Naltrexone (oral or monthly injection / Vivitrol)	Opioid receptor antagonist — blocks the endorphin reward signal from alcohol, reducing the 'wanting' and the reward	Strong — multiple RCTs; FDA approved	OPRM1 A118G carriers show dramatically superior response — up to 3x better outcomes. If you know you carry this variant, naltrexone is likely your highest-efficacy medication. Monthly injection (Vivitrol) removes the daily compliance variable. The Sinclair Method — taking naltrexone before each drinking episode to extinguish the reward response — is a validated harm reduction approach for those not ready for abstinence.
Acamprosate (Campral)	GABA and glutamate modulation — reduces the excitatory overdrive of alcohol withdrawal and PAWS; reduces distress of abstinence	Strong — FDA approved; particular efficacy in PAWS	Particularly relevant for the GABA-deficit profile (GABRA2 high-risk) and for those with severe PAWS. Renally cleared — no liver metabolism, which is an advantage in alcohol recovery where liver function may be compromised. Does not interact with the liver support stack. Start during or immediately after detox.
Disulfiram (Antabuse)	ALDH2 inhibition — blocks acetaldehyde clearance, producing severe nausea, flushing,	Moderate — aversion strategy; effective when compliance is	Works by making drinking immediately and severely punishing. Most effective when dosing is observed (partner, pharmacist, or

	and discomfort if alcohol is consumed	supervised	program). Not appropriate for ALDH2*2 carriers — already have impaired acetaldehyde clearance; disulfiram would intensify an already existing biological deterrent. Requires absolute commitment to abstinence as it makes drinking medically dangerous.
GLP-1 agonists (semaglutide, tirzepatide)	Reduce alcohol 'wanting' through dopamine and reward circuit modulation; 2025–2026 data shows 50–60% reduction in drinking episodes	Emerging — strong observational data; clinical trials ongoing as of 2026	The most significant development in alcohol treatment pharmacology in years. The mechanism appears to reduce the 'wanting' quality of alcohol craving — the compulsive pull toward the substance — rather than merely the reward once consumed. For patients with both AUD and metabolic concerns, this may address both simultaneously. Discuss with your physician.
Gabapentin	GABA-modulating anticonvulsant — reduces withdrawal symptoms and PAWS; particularly relevant for sleep and anxiety	Moderate — off-label; significant evidence base for AUD	Useful for PAWS management and sleep in the first 1–3 months. CAUTION: gabapentin itself carries dependence risk, particularly in people with addiction history. Use with time limits and physician monitoring. The GABRA2 high-risk profile may benefit most and carries highest misuse risk — monitor carefully.

A Final Word

THE CENTURION — The System That Chose Alcohol Was Not Broken. It Was Solving a Real Problem Badly.

The GABA system that could not quiet itself. The endorphin system that could not generate adequate social warmth. The dopamine system that could not find enough reward in the ordinary texture of daily life. The methylation system that could not synthesize sufficient serotonin. These are not character defects. They are biological vulnerabilities — specific, genetic, addressable.

Alcohol solved these problems. For years, perhaps for decades, it solved them. The solution was real. And it was destroying everything else in the process.

What the Recovery Kernel offers is a different kind of solution — slower, less dramatic, requiring daily practice and biological support and community and time. But a solution that does not cost the things alcohol was costing. That does not narrow your life down to the single question of when the next drink will come.

The brain volume lost to alcohol grows back. The GABA receptors recover. The serotonin system rebuilds. The liver heals. The relationships that were strained can be repaired — not all of them, and not quickly, but meaningfully. The cognitive function that alcohol was impairing returns, often surpassing the pre-drinking baseline because the tools built in recovery — the breathwork, the sleep, the nutrition, the exercise, the contemplative practice — are actively building a more capable brain than the one that started drinking.

The Recovery Kernel was built for people who needed a more precise map than the generic one being handed out. You now have the alcohol-specific version of that map.

The navigation continues in the main Compendium. Use it.

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Refer to the Recovery Kernel Complete Centurion Compendium for all referenced protocols.

Crisis: call or text 988 | Emergency: 911 | SAMHSA: 1-800-662-4357

⚠ HARM REDUCTION RESOURCE — NOT MEDICAL ADVICE